Identifying the Causal Effect of Political Regimes on Employment*

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Abstract

1 Introduction

The question studied here is whether political regimes, dichotomized as democracies and autocracies, affect the rate of growth of employment. But broader issues are at stake.

The central claim of "new institutionalism" is that institutions are the primary cause of economic development. The theoretical program has been laid out by North (1997: 224; italics supplied): "To make sense out of historical and contemporary evidence, we must rethink the whole process of economic growth.... The *primary source* of economic growth is the institutional/organizational structure of a political economy...." (For similar assertions, see Rodrik, Subramanian, and Trebbi 2002 and Acemoglu 2003). Yet the new institutionalism also recognizes that institutions are endogenous. As already North and Thomas (1973: 6) observed, "new institutional arrangements will not be set up unless the private benefits of their creation promise to exceed the costs."

The embarrassingly obvious thought is that if endogeneity is sufficiently strong, causal effects of institutions cannot be identified. Imagine that only some particular institutions exist under the given conditions. Then the effects of institutions cannot be distinguished from the effects of the conditions under which these institutions are found.

Consider the substantive question posed above in the context of the OECD countries. Since almost all of them had democratic regimes between 1950 and 1990 – the period studied here – it is not possible to

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determine whether the slow rate of growth of labor force in these countries, on the average 0.97 per annum as contrasted with 2.32 in the rest of the world, is the effect of democracy or of the high productivity of their labor force, on the average $19,257 per worker as opposed to $5,931 in other countries,[1] or perhaps of the already higher levels of participation, on the average 44 percent as compared to 40 percent elsewhere.

Whether the effect of political regimes can be identified in the world as a whole is the question pursued below. It is important, however, to keep in mind that identification is not a matter of sample size but of endogeneity. The reason causal effects of political regimes are next-to-impossible to identify among the OECD countries is not a small number of observations but the fact that history has mischievously eliminated autocracy in developed countries. The logic entailed in identifying causal effects does not depend on N (see Fearon 1991). Even if we are analyzing a single observation, we need to distinguish the effect of a cause from the effect of the conditions that activated this cause. Did the French revolution generate little social change, as Tocqueville (1964 [1856]) would have it, because revolutions result in little change or because they occur only in countries resistant to change?

The generic problem in identifying causal effects is how to answer the counterfactual question: what would have occurred had the cause been absent? But to engage in counterfactual inferences we need some systematic criteria to choose among several plausible candidates (Hathworn 1991). For example, the argument that colonialism had a positive effect on economic development of the colonies is based on the counterfactual hypothesis that these colonies would not have developed without foreign penetration, while claims that colonialism had a pernicious economic effect are based on the premise that they would have developed had they been left alone.[2] Whether we can successfully solve such problems is, in my view, largely a matter of luck, namely whether history has been kind enough to generate observations that can be used to inform us about the plausible counterfactuals. Hence, some causal effects may be identifiable, while other may not be.[3]

Since this is mainly a methodological paper, the theory is introduced rather briefly in Section 2, only to motivate the statistical model to be estimated. Section 3 emphasizes that to identify causal effects it is necessary to make assumptions about counterfactuals. Section 4 presents different biases that may be present due to non-random assignment ("se-

[1] All the dollar numbers are in 1985 purchasing power parity dollars from Penn World Tables, release 5.6.

[2] For a thoughtful discussion of this issue, see Kaniyathu (2006).

[3] For a more extensive discussion of these issues, see Przeworski (2006).

lection") of causes to exogenous conditions. Section 5 is a review of estimators designed to avoid some of these biases. In Section 6 these estimators are applied to the substantive problem at hand. Finally, Section 7 focuses on the effects of globalization.

2 Growth of Labor Force

Assume a Cobb-Douglas economy with constant returns to scale, of the form

$$Y_t = A_t F(K_t, L_t) = A_t K_t^{\alpha} L_t^{1-\alpha}. \tag{1}$$

The demand for labor in this economy is

$$L_t^* = \left(\frac{(1-\alpha)A}{w}\right)^{1/\alpha} K, \tag{2}$$

where w is the wage rate per unit of L. The rate of growth of labor force is thus given by

$$\frac{\dot{L}}{L} = \frac{\dot{K}}{K} + \frac{1}{\alpha}\left(\frac{\dot{A}}{A} - \frac{\dot{w}}{w}\right), \tag{3}$$

where the dots indicate time derivatives. One way to read this expression is that labor force grows at the same rate as the capital stock as long as increases in wages follow exactly the increases in Hicks-neutral productivity, $\dot{A}/A$. In turn, if wages grow slower than productivity, the growth of employment is faster than the growth of capital stock.

The average rate of growth of employment in the world between 1950 and 1990 is presented in Figure 1.[4]

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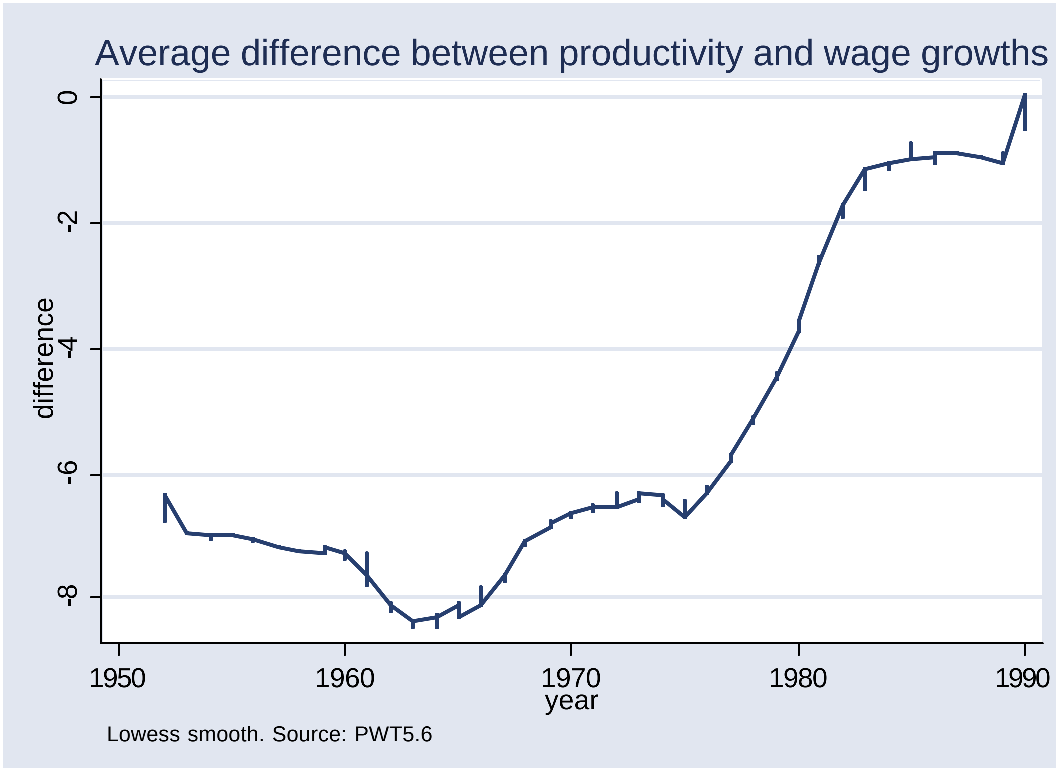
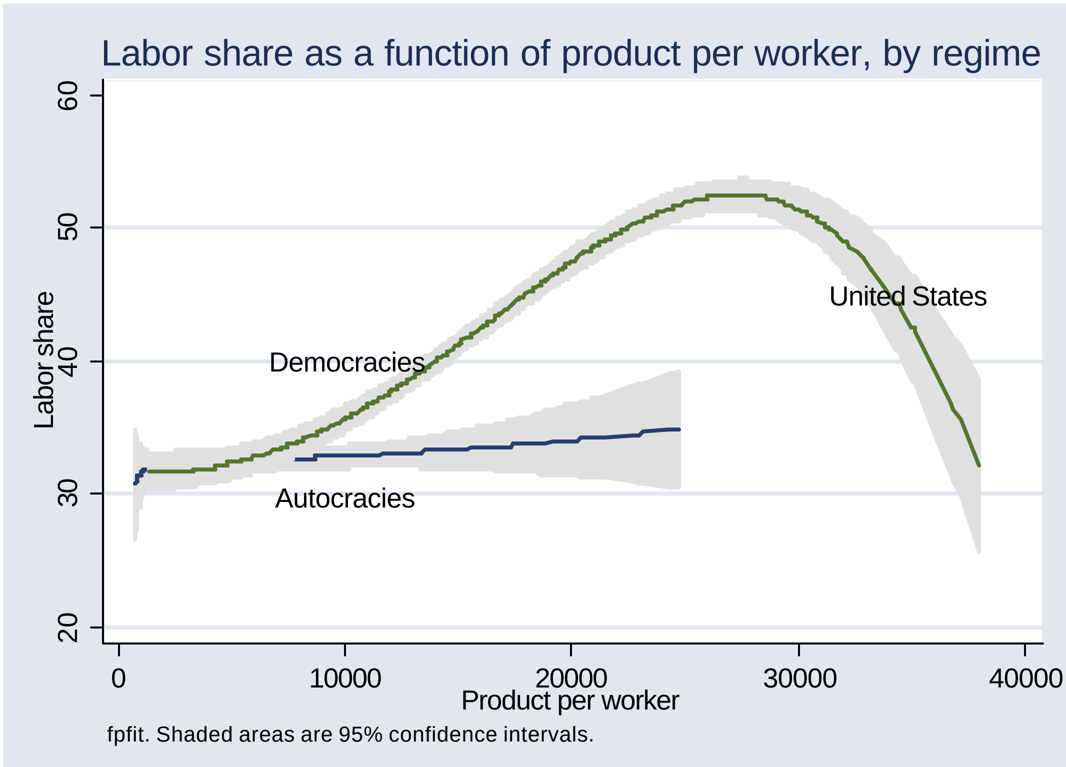
[4] The labor force series is obtained by dividing product per worker by product per capita from PWT 5.6. According to the ratings of data quality provided by PWT, the reliability of the product series varies greatly across countries. Przeworski et al. (2000: Appendix 3.1) calculated that this quality is much higher in democracies but did not find data quality to be a source of bias.

Given (3), we can use the series for labor force and capital stock to compare the rate of growth of productivity to that of wages. Figure 2 shows that until mid-1970s average increases of wages largely outpaced average increases of productivity, but this difference was rapidly reduced so that by 1990 wages and productivity grew at the same pace.



To introduce the effect of political regimes, assume that autocracies pay lower wages than democracies. The prima facie evidence for this assumption is based on Rodrik (1998) as well as Przeworski et al. (2000), who found that labor shares are lower in autocracies than in democracies at the same income levels. These data are reproduced in Figure 3.



The labor share data, however, are scarce, cover only the manufacturing sector, and are highly unreliable.[5] Hence, I will think in reduced form terms, assuming that the growth of wages is higher in democracies:

$$\frac{\dot{w}}{w} = \theta * REG, \tag{4}$$

$\theta > 0$, where $REG = 1$ if the political regime at time t is a democracy and $REG = 0$ otherwise. Letting $\frac{\dot{L}}{L} \equiv \gamma_L$, $\frac{\dot{K}}{K} \equiv \gamma_K$, and $\frac{\dot{A}}{A} \equiv \gamma_A$, we get

$$\gamma_L = \gamma_K + \frac{1}{\alpha}\gamma_A - \frac{\theta}{\alpha}REG, \tag{5}$$

where θ/α is the causal effect of political regimes on the growth of employment.

We seek to identify this causal effect using data from 135 countries between 1950 and 1990.[6] Assume that the rate of technical progress is

[5] Indeed, the World Bank stopped publishing them.

[6] This particular data set is used because this is the only period during which the information about capital stock is available. Economic data are from PWT5.6 and political data from Przeworski et. al. (2000).

constant over time (but it may vary across countries or across regimes). Then we can write (5) as

$$\gamma_L(it) = \beta_0 + \beta_1\gamma_K(it) + \beta_2 REG(it) + e(it), \tag{6}$$

where $\beta_0 = \frac{1}{\alpha}\gamma_A$ (or $\frac{1}{\alpha}\gamma_A(i)$), $\beta_1 = 1/\alpha$, and $\beta_2 = -\theta/\alpha$.

The question is whether it is possible to identify β_2 when regimes are endogenous.

3 The Problem

First, we need some notation. Let T stand for the (potential) cause, where $T = 1$ indicates "treatment" and $T = 0$ "control" (or a different treatment).[7] Without a loss of generality, we will think of democracy ($REG = 1$) as the treatment and of autocracy ($REG = 0$) as the control. X and V are "covariates," that is, traits of an individual unit prior to the application of the treatment. X is the vector of covariates observed by the researcher, V are covariates not observed. $Y = \{Y_0, Y_1\}$ is the variable subject to the potential effect of the cause, where Y_0 stands for states of the units not exposed to treatment and Y_1 of those exposed to it, so that for each unit i we observe either Y_1 or Y_0 :

$$Y_i = T_i Y_{1i} + (1 - T_i) Y_{0i}. \tag{7}$$

A "unit" is an opportunity for the cause to operate. It may be an individual, a country, or what not. Moreover, it may be the same individual or a country in a different state: say Sweden in 1950 and in 1951. Hence, the "unit" is a full set of observable and unobservable covariates: i is coextensive with the vector of "background conditions" $(\mathbf{x}_i, \mathbf{v}_i)$.

Now, what is the causal effect of treatment on the particular unit i, the Individual Treatment Effect? This effect is *defined* as the difference between the states of an individual unit when it is subjected and not subjected to the operation of the cause, in our case between the rate of growth of labor force of a particular country at a particular time under democracy and autocracy. Formally,[8]

$$ITE_i = y_{1i} - y_{0i} \equiv \beta_i \tag{8}$$

But even if the assignment of regimes to countries were random, this question could not be answered without making some assumptions about

[7] Although for simplicity I assume that the cause is a binary variable, everything said here holds for any discrete or continuous values of T.

[8] For simplicity, I will ignore time in the theoretical dicusssion.

hypothetical situations that would have occurred had a country that did not get treatment (had not been exposed to the potential causes) received it or had a country that did receive treatment not received it. Since these states did not occur, they are contrary to fact, *counterfactual*.[9] And since counterfactuals cannot be observed, assumptions about counterfactuals cannot be directly tested.[10] Hence, the effect of a cause on an individual unit cannot be determined without making assumptions about counterfactuals. These assumptions cannot be tested.

What assumption would identify the individual treatment effect under random assignment?

Assumption 1: *Unit homogeneity (Holland 1986).*

For any $i, j \in N$,

$$\textit{if } \{\mathbf{x}_i, \mathbf{v}_i\} = \{\mathbf{x}_j, \mathbf{v}_j\}, \textit{then } y_{0i} = y_{0j} \textit{ and } y_{1_i} = y_{1j}.$$

This assumption says that if any two units have the same values of covariates, they would have the same states under control and the same states under treatment. When this assumption is true, the process of selection can be ignored: it does not matter which of two identical units is subject to treatment and which serves as control.

This assumption *identifies* the causal effect of treatment. Assume that we observed i in state 1 and j in state 0. Applying the homogeneity assumption yields

$$ITE_i = y_{1,i} - y_{0,i} = y_{1,i} - y_{0,j},$$

where now both $y_{1,i}$ and $y_{0,j}$ are observed.

What does "*identify*" mean? Intuitively "to identify" is to be able to infer relations among variables (or the parameters of multivariate distribution) on the basis of all the possible observations (Koopmans 1949; in Manski 1995: 6). But very often this is possible only by assuming something that may or may not be testable. As Manski (1995: 18) observed, "Theories are testable where they are least needed, and are not testable where they are most needed. Theories are least needed to determine conditional distributions $P(y|x)$ on the support of $P(x)$. They are

[9] The idea of counterfactuals goes back to Pascal (1669, sec. 162): *"Le nez de Cléopâtre: s'il eût été plus court, toute la face de la terre aurait changé."* On the distinctions among different types of conditional propositions, see Edgington (2001). On the logical problems with counterfactuals, see Quine (1953), Lewis (1973), Mackie (2002 [1973]), Goodman (1979), and Stalnaker (1987).

[10] For a statistical view of causality without counterfactuals, see Dawid (2000), who rejects them as metaphysical.

most needed to determine these distributions off the support." We have seen that since each unit can be observed only in one state at one time it is not possible to identify the individual causal effect without making some assumptions. Hence, we need *identifying assumptions*, such as unit homogeneity. This assumption is not testable. But it seems reasonable.

Now we can ask about the Average Treatment Effect, ATE. Specifically, under what assumptions

$$\beta_{ATE} = E(Y_1 - Y_0|X) = E(\beta|X) = \bar{y}_1 - \bar{y}_0 = \bar{\beta},$$

so that the observed mean difference identifies the *average* treatment effect? The answer is "conditional mean independence":

Assumption 2: *Conditional Mean Independence.*

$$E(Y_1|X, T = 1) = E(Y_1|X, T = 0) = E(Y_1|X)$$

$$E(Y_0|X, T = 0) = E(Y_0|X, T = 1) = E(Y_0|X)$$

This assumption says that conditional on observed covariates we can expect the units not exposed to treatment to react to it identically to those observed under treatment and the units exposed to treatment not to differ in their control state from those observed under control.[11] Under random assignment this assumption is trivially true. And it implies that the observed difference identifies the average causal effect:[12]

$$\bar{\beta} = E(Y_1|X, T = 1) - E(Y_0|X, T = 0) = E(Y_1 - Y_0|X).$$

Hence, if the assignment to treatment is random, then the difference of the observed means identifies the average causal effect of treatment.

Now, let U stand for the effect of V on Y and assume linear separability. Then

$$E(Y|X, V) = E(Y|X) + U. \tag{9}$$

[11] To help with the notation, $E(Y_1|T = 1)$ is to be read as "the expected value of the outcome under treatment, given that the units have been observed as treated," while $E(Y_1|T = 0)$ as "the expected value of the outcome under treatment, given that the units have been observed as not treated."

[12] According to a theorem by Rosenbaum and Rubin (1983), if the conditional mean independence holds in the form specified in the text, then it also holds in the form in which $p(X) = \Pr(T = 1|X)$ is substituted for X, where $p(X)$ is the "propensity score."

Substituting into (7) (and dropping the i subscript) yields

$$\begin{aligned} Y = & E(Y_0|X) + T[E(Y_1 - Y_0|X)] + \{T(U_1 - U_0) + U_0\} = \\ & \beta_0(X) + \beta(X)T + U, \end{aligned} \tag{10}$$

where $\beta(X) = E(Y_1 - Y_0|X)$ is the average causal effect, discussed further below.

To identify the causal effect, we need to ensure that

$$U = T(U_1 - U_0)] + U_0 = 0,$$

where U is the impact of unobserved factors in $Y = \beta_0(X)+\beta(X)T+U$ and $\beta(X)$ is the average causal effect conditional on X. The basic concern in identifying causal effects is thus whether $E(U) = 0$.

4 Potential Biases

4.1 Baseline Bias

Note first that the causal effect of interest need not be the effect on the average unit but on those units that are actually observed as treated.[13] This *estimand* is typically referred to as the Average effect of Treatment on the Treated, ATT, defined as

$$\beta_{ATT} = E(Y_1 - Y_0|X, T = 1). \tag{11}$$

The value of this parameter tells us how the treatment changes the outcome for those unit that were observed as treated. Note that $E(Y_1|T = 1)$ is observed, while $E(Y_0|T = 1)$ is the missing counterfactual. Now consider the bias of the observed difference, $\bar{\beta}$, as an estimator of β_{ATT}:

$$\begin{aligned} \bar{\beta} - \beta_{ATT} = & E(Y_1|X, T = 1) - E(Y_0|X, T = 0) - E(Y_1 - Y_0|X, T = 1) \\ = & E(Y_0|X, T = 1) - E(Y_0|X, T = 0) \\ = & E(U_0|T = 1) - E(U_0|T = 0), \end{aligned} \tag{12}$$

where the last expression is the difference in the control state between those units that were treated and those that were not, typically referred

[13] This effect is of particular interest in remedial policy programs. As Heckman repeatedly points out, it makes no sense to ask what would be the effect of manpower training program on millionaires. In turn, we want to know the effectiveness of such programs for the people who need them and get them.

to as the "baseline bias." Suppose, for example, that an omitted variable, say human capital, H, is correlated with the treatment and it affects the employment prospects of a country, so that $E(U_0|H = high, T = 1) > E(U_0|H = low, T = 0)$. Since countries in countries observed under $T = 1$ employment would have grown faster under $T = 0$ than those actually observed under $T = 0$, the observed difference overestimates the causal effect of T. This bias is sometimes referred to as "the" selection bias, but we will see that there are other potential selection biases than the baseline bias.

4.2 Self-Selection Bias

Now, return to ATE. The bias of $\bar{\beta}$ as the estimator of β_{ATE} is

$$\bar{\beta} - \beta_{ATE} = E(Y_1|X, T = 1) - E(Y_0|X, T = 0) - E(Y_1 - Y_0|X). \quad (13)$$

Adding and subtracting $E(Y_0|T = 1)$ yields

$$\begin{aligned} \bar{\beta} - \beta_{ATE} = \{E(Y_0|X, T = 1) - E(Y_0|X, T = 0)\} + \\ \{E(Y_1 - Y_0|X, T = 1) - E(Y_1 - Y_0|X)\} = \\ \{E(U_0|T = 1) - E(U_0|T = 0)\} \\ + \{E(U_1 - U_0|T = 1) - E(U_1 - U_0)\}. \end{aligned} \quad (14)$$

The term in the first curly brackets is the by now familiar baseline bias. The term in the second brackets, in turn, is best thought of as "self-selection" bias. This term is the difference between the effect of treatment on those who were actually treated and on the average unit. But why would the effect of the treatment on the treated differ from its effect on those who are not? One reason is that recruitment to treatment depends on something not observed by the researcher but anticipated by the unit. This will occur if units seek treatment for some reasons other than the $X's$ observed by the researcher or if they comply differently with the treatment depending on the $X's$. Suppose – I am not asking you to believe it – that political elites which opt for democracy also know how to make employment grow faster. Then the effect of democracy on the growth of employment for the countries observed as democracies will differ from the effect on the average country: a self-selection bias.

4.3 Post-treatment Bias: "Manipulability" and "Attributes"

Thus far we have assumed that the $X's$ and the $V's$, called here "covariates," do not change with treatment. The assumption was that causes can be manipulated one-at-a-time. But suppose that some of the covariates – call this subset A for "attributes" – change as the effect of treatment: this is called "post-treatment effect" by King and Zeng (2002). Now the treatment may have two effects: a direct one and an indirect via A. We need some identification assumptions to tell these two effects apart.

Can we always make such assumptions? Here we enter into a complex and subtle issue. According to Holland (1986), to qualify as a potential cause, the particular variable must be vulnerable to (potential) *manipulation.* The critical feature of the notion of cause is that different values of the cause can be realized under the same background conditions. This is why attributes, such race or gender, cannot be causes. "Causes," Holland says, "are only those things that could, in principle, be treatments in experiments" (1986: 954). What distinguishes statistical association from causation is manipulability: "the schooling a student receives can be a cause, in our sense, of the student's performance on a test, whereas the student's race or gender cannot." It makes no sense to say "Joe earns \$500 less than Jim *because* Joe is black," since skin color (called "race" in the United States) cannot be manipulated. Causal inference is concerned with the effect of causes under specific background conditions ("on specific units") and attributes cannot be manipulated without changing these conditions.

This arguments confounds two propositions: (1) T cannot be manipulated and (2) T cannot be manipulated without changing A. The first one says that we cannot change the skin color of an individual. The second says that we can change it but if we change it, we will also change other characteristics of this individual (or the treatment of this individual by others). The confusion becomes apparent when we read that "An attribute cannot be a cause in an experiment, because the notion of *potential exposability* does not apply to it. The only way for an attribute to change its value [so it can be changed!] is for the unit to change in some way and no longer be the same unit" (Holland 1986: 954). Now, if (1) holds, it may still be true that there are other units that have the same background conditions but a different value of T and we can use the conditional mean independence assumption to identify the causal effect. Only if (2) is true, does identification become impossible.

Consider an example closer to our practice: the location of a country in Africa, which in many analyses appears to affect civil strife and economic growth. Does it make sense to say that "the effect of Africa on growth is β"? "Africa" is clearly an attribute by Holland's definition, a set of related unobserved characteristics. If history had placed Zimbabwe in Latin America, it would have no longer been Zimbabwe: it would differ in various ways that make Africa distinct from Latin America. Hence, relying on the Africa dummy to generate counterfactuals would generate a "post-treatment bias."

King and Zeng (2002: 21) emphasize that controlling (matching) for variables that are endogenous with regard to treatment generates bias. This can be seen as follows. For simplicity, suppose that assignment is random, so that there is no baseline or self-selection bias, but $X_1 = X_0 + \delta T$. Then conditioning on X,

$$E(Y_1 - Y_0|X) = E(Y_1|X_0 + \delta T) - E(Y_0|X_0) = E(Y_1 - Y_0|X_0) + \{E(Y_1|X_0 + \delta T) - E(Y_1|X_0)\}, \tag{15}$$

where the last term is the "post-treatment bias." For example, suppose that capital stock grows slower under dictatorships. Conditioning on the growth of growth of capital stock would then generate post-treatment bias.

4.4 Non-independence Bias: "SUTVA"

One final implicit assumption concerns independence of the Y variables across units. This assumption is called SUTVA, for "stable unit treatment value." Suppose that the units are individuals and that they learn from one another, so that $y_i = f(y_j)$. This means that the performance of the treated may affect the performance of the untreated, or vice versa. In Lucas's (1988) growth model, a young plumber learns from the experienced one. Hence, if we take the difference in their productivity as the effect of experience, it will be underestimated because of the externality. Or take T to be "export-oriented" strategy. South Korea adopted this strategy early and had high growth rates. Brazil adopted it late. But suppose that Brazil had adopted it early: would the growth rate of Korea been the same? If it would not have been the same, the values observed for Korea under treatment depend on the realization of the treatment variable for Brazil: hence the Korean values are not "stable."

In our context, this assumption is particularly dubious. In an open economy, the rate of growth of employment in one country depends on its growth in other countries. Hence, if country i that is an autocracy in which wages grow slowly and employment quickly (say China) were to become a democracy in which employment would grow slower, the rate of growth of employment in other countries would accelerate. One needs some kind of a world equilibrium model to identify the causal effect when this assumption is violated.

5 Types of Estimators

How can we identify causal effects when the assignment is not random?[14] Basically, we can adopt two approaches: drop the observations that are not "comparable," restricting identification of causal effects to those that are, or keep all the observations and generate hypothetical matches for each of them. Matching procedures would eliminate (or give almost zero weights to) all the observations that do not have close matches, while procedures generating hypothetical counterfactuals would fill all the growth cells for which history did not generate the information.

5.1 Matching

One way to proceed is to *match on observables.*[15] Say we want to examine the effect of guaranteed income programs on labor supply. We observe some wealthy countries with such programs (*Revenue minimum d'insertion* in France) and many countries, rich and poor, without them. We would not want to match the wealthy treatment cases with controls from poor countries. Hence, we use as controls countries with comparable per capita income, and restrict our causal inference to such countries.

Matching takes the assignment of causes as given and calculates causal effects conditional on the assignment of causes realized by history, relying on the conditional mean independence assumption

$$E(Y_j|X, T = j) = E(Y_j|X) \forall j, \tag{16}$$

which says that the value of Y in any state j does not depend on the state T in which a unit is observed once it is conditioned on the observed covariates. This is the same assumption as conditional mean independence introduced above, but written more generally to emphasize that the cause may assume any set of values.

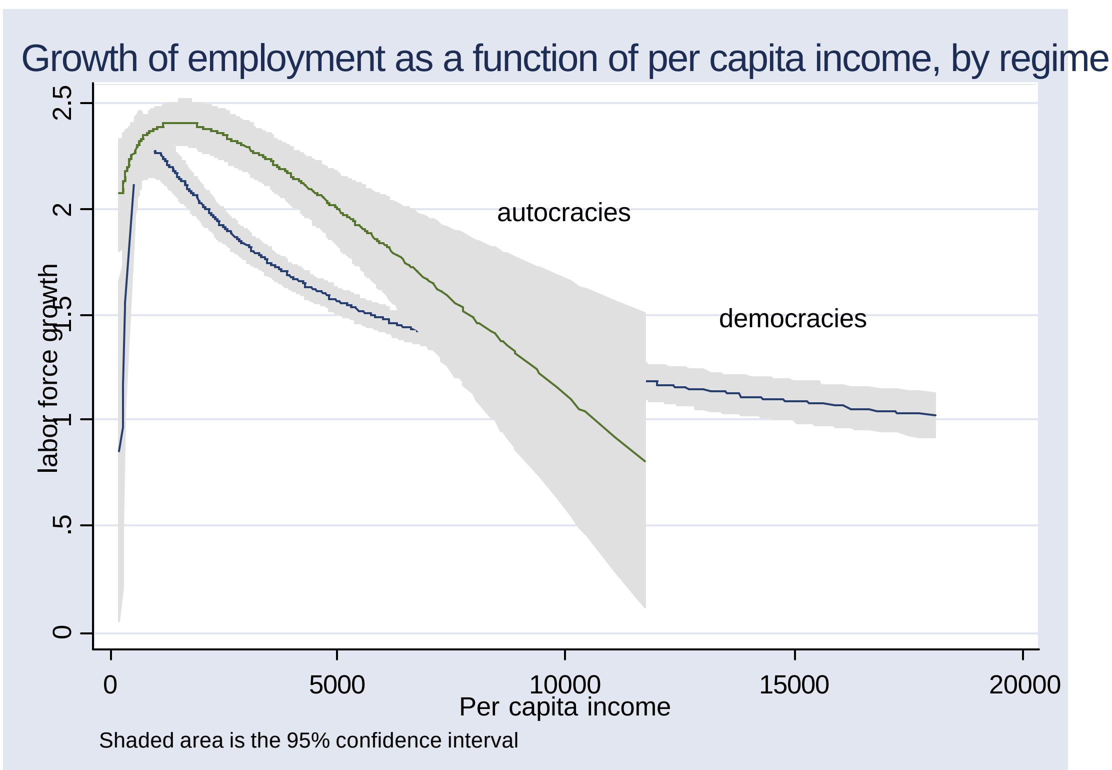
Matching estimators are vulnerable to two problems:

(1) Dropping observations reduces the scope of generality. Sometimes, as in the example of minimum income programs, this is not a loss. It is not a loss because the probability that a poor country would institute these programs is zero: poor countries cannot afford such programs, so that the question how these programs would affect labor supply in poor countries is moot. But how should we proceed when this

[14] For overviews of estimators see Angrist and Krueger (1999), Berk (2004, Chapter 5), Du- o (2002), Persson and Tabelini (2003, Chapter 5), or Winship and Morgan (1999). For reasons of space, I do not discuss difference-in-difference estimators, for which see Woolridge (2002) and Bertrand, Duflo, Mullainathan (2004).

[15] On matching estimators, see Rosenbaum (2002), Imbens (2002), Becker and Ichino (2002), and, more critically, Heckman (2004).

probability is positive under all conditions, yet very differently distributed with regard to these conditions, as in the case of political regimes? What to do with observations without a close match? Figure 1 shows a semi-parametric (fractional polynomial) regression of labor force growth on per capita income, by regime. Since some democracies are located in the range where there are no autocracies, different matching algorithms will either drop these observations of democracies or assign to them very low weights.[16] In either case, we have to worry whether the causal effect is the same for those observations with close matches and those without them. Moreover, as King and Zeng (2002) emphasize, extrapolations out of range of common support are highly sensitive to the form of the function.



(2) We can match on observables. But should we not worry about unobservables? Suppose that leaders of some countries go to study in

[16] Depending on the algorithm, matching estimators treat differently observations that cannot be matched exactly. When matching is restricted to common support or when it is confined to balanced strata, observations without a match are ignored. When some kind of distance measure is employed, distant matches obtain weights approaching zero.

Cambridges, where they absorb the ideals of democracy and learn how to promote employment. Leaders of other countries, however, go to the School for the Americas, where they learn how to repress and nothing about economics. Autocracies will then generate lower growth because of the quality of the leadership, which is not observed. Since this is a variable we could not observe systematically, we cannot match on it. And it may matter. Conditional mean independence – the assumption that unobserved factors do not matter – is very strong, and likely to be often false in cross-national research.

All that was said about matching applies to regression models that control for the observables. Matching is just a non-parametric regression: both generate means of Y conditional on X and T. Moreover, as observed respectively by Manski (1995) and Achen (1986), both matching and parametric regressions that control for observables may in fact exacerbate the biases due to selection on unobservables.

Both matching and parametric regression estimates can be subjected to sensitivity analysis. Given assumptions about the unobservables, one can calculate the range of estimates that are compatible with the observed data (Manski 1995). Rosenbaum (2002, Chapter 4) presents methods for quantifying the sensitivity of the estimates of causal effects under different assumptions. Obviously, the more plausible the assumption and the narrower the bounds, the more credible is the estimate.

5.2 Instrumental Variables

Instrumental variables estimator is based on the assumption of conditional mean independence in the form:

$$E(Y_j|X, Z, T = j) = E(Y_j|X, Z)\forall j. \tag{17}$$

The idea is the following. Suppose that after conditioning on X, Y_j still depends on j, in other terms that $cov(T, U) \neq 0$. Now, suppose that there is a variable Z, called an "instrument," such that

$$cov(Z, T) \neq 0 \tag{18}$$

and

$$cov(Z, U) = 0. \tag{19}$$

Then conditioning on X and Z satisfies (11). Thinking in regression terms, let $\hat{Y} = f(Z)$ and $\hat{T} = g(Z)$. Then, by assumption (17), β in $\hat{Y} = \beta\hat{T}$ is that part of the causal effect of T on Y which is independent of U.

To qualify as an instrument, a variable must be related to the cause and only to the cause, so that its entire effect is transmitted by the cause. Note that while the assumption that the instrument is related to the cause (conditional on all exogenous variables) can be and should be tested, the assumption that it is independent of the conditions that also shape the effect is not testable.

Instruments must be correlated with the cause. Weak instruments (those weakly correlated with the treatment) can generate biased estimates even with very large samples. But instruments cannot be too strongly correlated with the cause. In the limit, if the instrument and the cause are the same, the instrument is as endogenous as the cause: this is "the curse of strong instruments." The causal effect cannot be identified, because it is impossible to separate the impact of the cause from that of the conditions that give rise to it.

In turn, the "exclusion restriction" (19) requires that the instrument have no effect that is not mediated by the cause. Moreover, given that $U = T(U_1 - U_0) + U_0$,

$$cov(Z, U) = cov(Z, U_0) + cov(Z, T(U_1 - U_0)). \tag{20}$$

Hence, the exclusion restriction has two parts, and Heckman (1996, 2004) repeatedly makes the point that, even if $cov(Z, U_0) = 0$, in the presence of unobserved self-selection the second covariance will not be zero.

5.3 Selection on Unobservables

Both matching and instrumental variables estimators condition on observed covariates and both are vulnerable to the influence of unobserved variables that are correlated with the treatment. Another approach conditions on unobserved as well as on observed covariates. One way to think of these estimators is that they emulate experiments, but differently than matching: not by eliminating observations that do not have an observed match but by creating observations to match all the observed values. The assumption is that if the conditioning is correct, then the resulting data have the same structure as if history had performed a random experiment assigning different values of treatment to each unit. Since the conditional mean independence of the form

$$E(Y_j|X, Z, V, T = j) = E(Y_j|X, Z, V)\forall j \tag{21}$$

holds whenever assignment is random, the only issue with regard to these estimators is whether they correctly emulated random assignment.

The basic idea is the following. We first describe the process by which the observed assignment of causes was generated by history:

$$T^* = Z\alpha + V, T = 1(T^* > 0), V \sim (0,1). \tag{22}$$

This equation says that the propensity toward being observed under treatment depends on observable variables Z and unobserved factors V and that we observe $T = 1$ if $T^* > 0$.

Secondly, we exploit the possibility that $cov(V,U) \neq 0$, by expressing $E(U_j|T = j)$ in

$$E(Y_j|X, T = j) = E(Y_j|X) + E(U_j|T = j), \tag{23}$$

as

$$E(U_j|T = j) = \theta_j E(V|T = j), \tag{24}$$

where the latter expectation can be estimated from (22). Finally, we substitute, to obtain

$$E(Y_j|X, T = j) = E(Y_j|X) + \theta_j E(V|T = j), \tag{25}$$

which can be now estimated by least squares. The OLS coefficients of $E(Y_j|X) = X\beta_j$ can be then used to generate counterfactual values of Y_j for the cases in which it is not observed, thus filling all the missing matches. Finally, for $j = \{0, 1\}$,

$$\hat{\beta}_{ATE} = E(Y_1|X) - E(Y_0|X) = (\hat{\beta}_1 - \hat{\beta}_0)X,$$

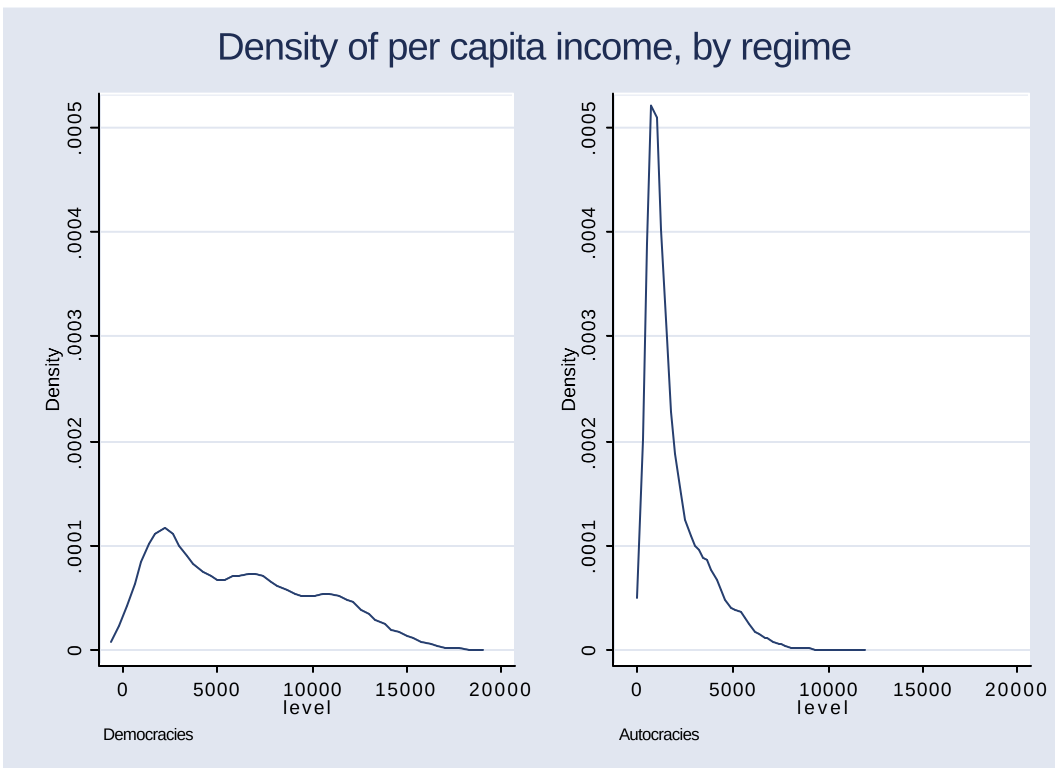
is the estimator of the average causal effect.

Note that we still have to be concerned about strong endogeneity of treatment. In principle, it has to be true that $0 < \Pr(T = 1|Z) < 1 \forall Z$. Otherwise, the counterfactuals cannot be realized given the mechanism by which history assigns treatments, so that the entire exercise is moot. The main vulnerability of this class of estimators stems from the untestable assumption about the joint distribution (V, U_1, U_0).

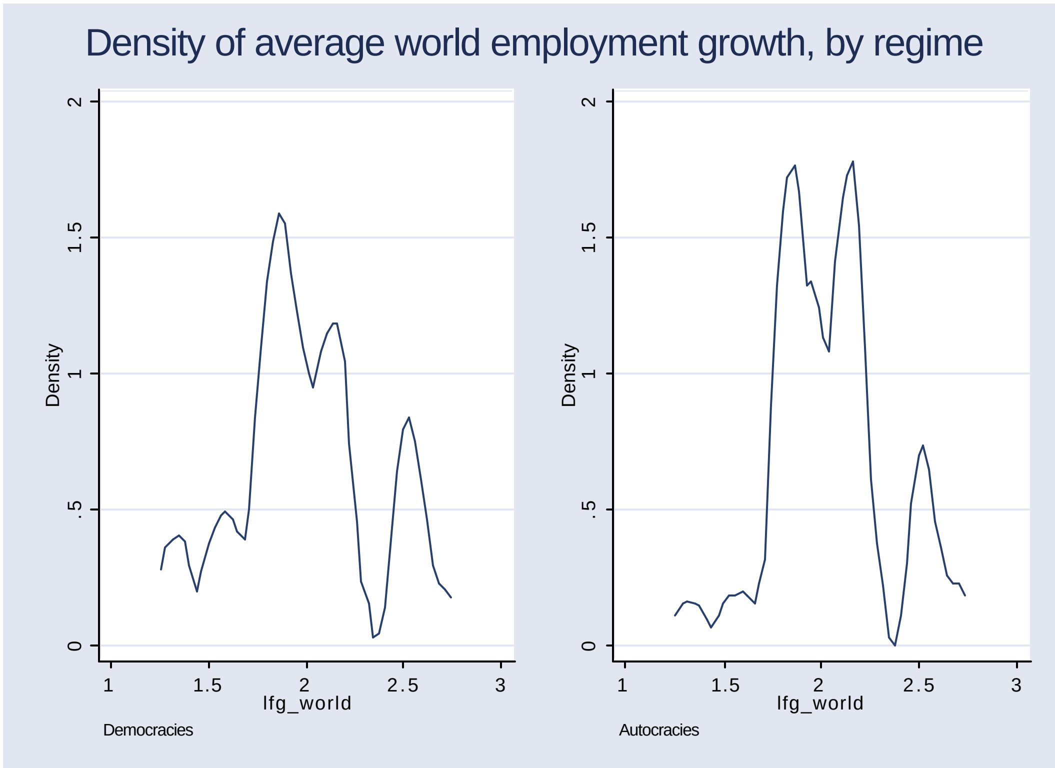
6 Political Regimes and the Growth of Employment

With this background, we return to the effect of political regimes on the growth of employment between 1950 and 1990. We observe 1595 democratic years and 2396 autocratic ones. The mean rate of growth of labor force under democracy was 1.59 (s.d.=1.20) and under autocracy 2.28 (s.d.=1.94), for a difference of -0.69. Obviously, comparing means is the same as regression, so it generates the same result, with a standard error of 0.05.Yet we already know that the difference of observed means is a biased estimator of the average causal effect if the assignment of regimes is not random, and it is easy to see that it is not.

Figure 4 shows the density of per capita incomes by regime. As one would expect, autocracies tend to be poor, while democracies can be found at all income levels. Indeed the wealthiest autocracy in the data set, Singapore in 1990 with per capita income of $\$11,698$, was poorer than 200 years of democracies, with the US in 1989 leading the list at $\$18,095$ in 1989.



Moreover, Figure 5 shows that democracies were somewhat more frequent during the years when the average rate of growth of labor force in the world was lower.



To identify the causal effects of regimes, we must therefore, distinguish it from the effect of the conditions under which these regimes were found. To do so, we will augment the theoretically derive specification given in (6) by adding some controls. These include the lagged proportion of the population that is employed (since labor force cannot grow when everyone is employed), the average rate of growth of labor force in the world during a particular year (as a crude attempt to take into account the world equilibrium effects), and per capita income (as a crude attempt to control supply effects, on the assumption that preference for leisure increases in income). Hence, we will be estimating models of the form:

$$lfg = \beta_0+\beta_1 ksg\_lag+\beta_2 lfprop\_lag+\beta_3 lfg\_world+\beta_4 level+\beta_{ATE}REG+e, \quad (26)$$

where lfg stands for $l$abor $f$orce $g$rowth, ksg_lag is the lagged value of $c$apital $s$tock $g$rowth,[17] $lfprop\_lag$ is the lagged value of $l$abor $f$orce

[17] Przeworski et. al. (2000) performed various tests and found that the growth of capital stock is exogenous with regard to regimes.

proportion in the populaton, lfg_world is the mean rate of growth of labor force in the world in a particular year, and $level$ is income per capita. The parameter of interest is β_{ATE}. The Appendix contains the STATA output of estimating this equation by OLS with pooled data, OLS with country clustered standard errors, and panel corrected standard errors (with country specific autocorrelation and the pairwise option). With all the controls, β_{ATE} ranges from -0.27 to -0.25 and it is always significant. Since these estimates are biased if selection is not random, they serve as a benchmark.

In addition, in the instrumental variables and the selection on unobservables models, we will augment (26) by a selection equation of the form

$$\Pr(REG = 1) = \Pr(Z\alpha + V > 0) = F(Z\alpha). \tag{27}$$

The probit model included in the Appendix uses the specification based on Przeworski et. al. (2000). It includes per capita income ($levlag$), proportion of countries in the world that are democracies in a particular year ($odwp$), the number of completed spells of democracy in the history of the country ($stra$), where all these values are lagged one year, and interactions of these variables with the lagged regime (autocracy=1).

The results are surprisingly robust.[18] Here is a table that summarizes the estimates of β_{ATE}:

Table 1: Different estimates of the average treatment effect.

$Estimator$	β_{ATE}	$s.e.$	t	p
OLS^a	-0.27	0.110	-2.56	0.010
$Fixed\ effects$	-0.15	0.093	-1.63	0.104
$Match-Kernel^b$	-0.29			
$Match-Neighbor^b$	-0.30			
$Match-Strata^b$	-0.29			
$Match\_Imbens^c$	-0.18	0.074	-2.44	0.015
$Heckman-two^d$	-0.26	0.015	-17.33	0.000
$Heckman-all^e$	-0.29	0.070	-4.14	0.000
$2SLS^f$	-0.29	0.070	-4.13	0.000

[18] The parametric estimators are not sensitive to the specification of the selection equation but matching estimates become lower when the variable $stra$ is dropped from this equation.

Notes: *a* Panel corrected standard errors. Other OLS results are similar (see the Appendix). *b* Standard errors are not given for these matching estimators since the average treatment effect was calculated as the weighted average of ATT and ATC (see below). *c* Imbens nnmatch with 5 matches. *d* Heckman two-steps estimator, with separate regressions for each regime. *e* Heckman estimator with all the observations considered together. *f* With propensity score as the instrument. Using all the instruments separately generates an almost identical result.

Similar conclusions apply to the estimates of the effect of the treatment on the treated (ATT) and the effect of the treatment on the control (ATC). Since we took the treatment to be democracy, the first estimates tell us what would have been the difference in the growth of employment for the units observed as democracies had they been autocracies under the identical conditions, while the second inform us what would have been the difference for the countries observed as autocracies had they been democracies (The signs are inverted to facilitate the interpretation.)

Table 2: Estimates of the effect of the treatment on the treated and on the control group.

Estimator	*Democracies* β_{ATT}	*as* *s.e.*	*Autocracies* *z*	*Autocracies* β_{ATC}	*as* *s.e.*	*Democracies* *z*
Kernel	0.43	0.11	3.93	−0.20	0.05	−3.62
Neighbor	0.48	0.49	0.98	−0.19	0.12	−1.59
Strata	0.39	0.08	4.74	−0.23	0.06	−3.92
Heckman	0.44	0.02	27.7	−0.02	0.03	−0.81

These estimates indicate that countries observed as democracies (which tend to be more developed) would have had a much faster employment growth had they been autocracies. In turn, the countries observed as autocracies would have had a somewhat slower employment growth as democracies, but the difference is about a half of that for the observed democracies.

Thus from the methodological point of view, this exercise turned out to be disappointing. There appear to be no selection biases with regard the growth of labor force, so that all the estimators generate similar results.[19] It seems safe to conclude that the growth of employment is somewhat slower in democracies by some amount between 0.15 and 0.30.

[19] In some other contexts, different estimators generate highly disparate conclusions. See Przeworski (2006).

7 Globalization

Yet all the estimators we used are based on the assumption of stable unit treatment value, which is unlikely to be satisfied in a globalized economy. Note that all the parametric analyses indicate that the rate of growth of labor force in each country is positively affected by its rate of growth in the world as a whole. Suppose that a country experienced a transition to democracy. Since employment grows slower in democracies, the world average would become lower, thus affecting the rate of growth in each country. This is clearly a violation of SUTVA, even if the bias it generates may be low.

To analyze the consequences of the mobility of capital and of commodities, one would have to study a world economic equilibrium, which I will not do. Instead, we can analyze the effect of the average growth of employment in autocracies in a particular year on the growth of employment in each of the democracies during this year.

The estimates are again robust for all parametric models (different versions of OLS, IV, and Heckman), so I do not enter into details. Reestimating all the parametric models with the effect of the mean growth of employment in autocracies increases the estimate of the effect of democracy on the growth of employment from a ball park number of -0.27 to about -0.43. With only minor variations, these analyses show that (1) labor force in democracies grows when employment increases in autocracies, indicating that autocracies and democracies respond similarly to fluctuations in world demand, but (2) this effect interacts negatively with per capita income. Since (using estimates from Heckman-two) the effect of growth in autocracies on the growth in democracies is $+0.52$ and the effect of interaction with per capita income of each of the democracies is -0.0393 per thousand, in a poor democracy like India, with per capita income of about $\$1,000$, the rate of growth of labor force increases by 0.48, while in a wealthy democracy such as the United States, with an income of $\$18,000$, employment growth slows down by about 0.19. when employment in autocracies increases by 1%. Democracies with per capita income of $0.52/0.0393 = 13,343$, the income of Switzerland in 1971, neither benefit nor lose when employment in autocracies increases. Note that in 1990 the unweighted average of capita incomes in the OECD countries was $\$13,650$. Thirteen countries, with incomes equal or higher to that of Iceland were net employment losers because of competition with autocracies.

Here, then, is the story: At each income level, capital stock grows at about the same rate in the two regimes. Yet autocracies repress wages; hence, they employ workers with lower marginal product; hence, their employment grows faster. In turn, this implies that democracies employ only workers with higher marginal product; hence, their employment grows slower. Since, as Figure 1 shows, the gap in wages opens up with per capita income, the effect of competition with autocracies is greater in the more developed democracies.

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